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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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Mark B. Wilson
FULBRIGHT & JAWORSKI L.L.P.
Suite 2400
600 Congress Avenue
Austin, TX 78701

EXAMINER	
CROUCH, DEBORAH	
ART UNIT	**PAPER NUMBER**
1632	

DATE MAILED: 11/29/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/943,984	HUNG ET AL.	
	Examiner	Art Unit	
	Deborah Crouch, Ph.D.	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 June 2004*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *76-189* is/are pending in the application.

4a) Of the above claim(s) *76-105,122-149,188 and 189* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *106-121 and 150 –187* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *31 August 2001* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *2/25/02*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

The amendment filed June 24, 2004 has been entered. Claims 76-189 are pending. Claims 76-105, 122-149, 188 and 189 are withdrawn from consideration. Claims 106-121 and 150 -187 are examined in this office action.

Applicant's election without traverse of claims 76, 106-121 and 150-187 in the reply filed on June 24, 2004 is acknowledged. Applicant's statements regarding allowance or the generic claim are acknowledged.

A rejection based on double patenting of the "same invention" type finds its support in the language of 35 U.S.C. 101 which states that "whoever invents or discovers any new and useful process ... may obtain a patent therefore ..." (Emphasis added). Thus, the term "same invention," in this context, means an invention drawn to identical subject matter. See *Miller v. Eagle Mfg. Co.*, 151 U.S. 186 (1894); *In re Ockert*, 245 F.2d 467, 114 USPQ 330 (CCPA 1957); and *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970).

A statutory type (35 U.S.C. 101) double patenting rejection can be overcome by canceling or amending the conflicting claims so they are no longer coextensive in scope. The filing of a terminal disclaimer cannot overcome a double patenting rejection based upon 35 U.S.C. 101.

Claims 173 and 184 are rejected under 35 U.S.C. 101 as claiming the same invention as that of claims 35-38 of prior U.S. Patent 6.395,712. This is a double patenting rejection.

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970);and, *In re Thorington,* 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 76, 106-121 and 150-187 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 18-33 and 37-43 of U.S. Patent No. 5,641,484 ('484) in view of

Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322. Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims are obvious over claims 18-33 and 37-43 of '484 in view of chemotherapeutic agents known in the art at the time of filing.

Claims 76, 106-121 and 150-187 are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 18-33 and 37-43 of '484 are drawn to methods to suppress the growth of a neu oncogene mediated tumor of a mammal comprising introducing to said tumor either a vector or a liposome comprising a nucleic acid encoding an SV40 large T antigen and/or a vector or a liposome comprising a nucleic acid encoding an E1A gene product, a liposome composition comprising a lipid complexed to a DNA segment encoding an E1A gene product.

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibits tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

At the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121 and 150-187 given claims 18-33 and 37-43 of '484 in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

Claims 76, 106-121 and 150-187 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-10, 21 and 22 of U.S. Patent No. 5,643,567 ('567) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994)

Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322. Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims are obvious over claims 1-10, 21 and 22 of '567 of '567 in view of chemotherapeutic agents known in the art at the time of filing.

Claims 76, 106-121 and 150-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 1-10, 21 and 22 of '567 are drawn to methods to suppress the growth of a neu oncogene mediated tumor, a method of providing to an adenovirus E1A gene product to a neu oncogene mediated tumor in a mammal and a method to suppress the growth of a of neu oncogene mediated tumor, comprising introducing to a cell of the tumor an adenovirus containing an adenoviral E1A gene operatively linked to a promoter.

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibits tumor growth in

patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

At the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121 and 150-187 given claims 1-10, 21 and 22 of '567 in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

Claims 76, 106-121 and 150-187are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-8 of U.S. Patent No. 5,651,964 ('964) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322. Although the conflicting claims are not identical, they are not patentably distinct from each other because

the present claims are obvious over claims 1-8 of '964 in view of chemotherapeutic agents known in the art at the time of filing.

Claims 76, 106-121 and 150-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claim 1-8 of '964 are drawn to methods to suppress the growth a neu oncogene-mediated tumors comprising introducing to the tumor a vector comprising a nucleic acid sequence encoding an adenoviral E1A gene product operatively linked to a promoter, where the vector is an plasmid, an adenovirus or an retrovirus.

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibits tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col.

2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

At the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121 and 150-187 given claims 1-8 of '964 in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

Claims 76, 106-121 and 150-187are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-32 of U.S. Patent No. 5,814,315 ('315) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322. Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims are obvious over claims 1-32 of '315 in view of chemotherapeutic agents known in the art at the time of filing.

Claims 76, 106-121 and 150-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where

the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 1-32 of '315 are drawn to methods to suppress an oncogenic phenotype in a neu oncogene overexpressing cell in a mammalian tumor by introducing to said tumor a nucleic acid encoding an adenoviral E1a gene product operatively linked to a promoter, the nucleic acid being either part of an adenovirus or viral vector, or the nucleic acid being complexed with a lipid component.

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibits tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

At the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121 and 150-187 given claims 1-32 of '315 in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

Claims 76, 106-121 and 150-187are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-24 of U.S. Patent No. 6,197,754 ('754). Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims are obvious over claims 1-24 of '754 in view of chemotherapeutic agents known in the art at the time of filing.

Claims 76, 106-121 and 150-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 1-24 of '754 are drawn to methods to suppressing growth of a tumor comprising introducing an mini-E1A gene product to a tumor and where the method further comprises introducing a chemotherapeutic agent.

At the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121 and 150-187 given claims 1-24 of '754.

Claims 76, 106-121 and 150-187are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-28 of U.S. Patent No. 6,683,059 ('059) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J. Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322. Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims are obvious uses of the products claimed in '059.

Claims 76, 106-121 and 150-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 1-28 of '059 are drawn to a nucleic acid encoding a mini-E1A gene product, a vector comprising a nucleic acid segment encoding an mini-E1A gene products, a mammalian cell comprising the nucleic acid vector and a non-viral gene delivery complex comprising a mini-E1A gene product.

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibits tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

At the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121 and 150-187 given claims 1-28 of '059 in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J . Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322. The present claimed methods are obvious uses for the products of claims 1-28 of '059.

Claims 76, 106-121,150-172, 174-183 and 185-187are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-34 and 42 of U.S. Patent No. 6,395,712 ('712). Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims are species to the genus claims in '712.

Claims 76, 106-121,150-172, 174-183 and 185-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product, expressing the E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 1-34 and 42 of '712 are drawn to a method for suppressing growth of a tumor comprising a neu oncogene containing cell comprising introducing a nucleic acid encoding a an E1A gene product using a DNA/liposome complex and contacting the cell with cisplatin or paclitaxel, a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and cisplatin or paclitaxel.

The present claims require expression of the nucleic acid making them species to claims 1-34 and 42 in '712, which do not require expression. Further, the present claims are genus to claims 1-34 and 42 in '712 because

the specific agents claimed are species to the genus of agents presently claimed. Therefore, at the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121,150-172, 174-183 and 185-187 given claims 1-34 and 42 of '712.

Claims 76, 106-121,150-172, 174-183 and 185-187are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-54 of U.S. Patent No. 6,326,356 ('356). Although the conflicting claims are not identical, they are not patentably distinct from each other because the present claims encompass the nucleic acid as encoding an mini-E1A gene as claimed in '356.

Claims 76, 106-121,150-172, 174-183 and 185-187are drawn to methods for suppressing growth of a tumor comprising a neu oncogene cell, comprising contacting the cell in the tumor with a nucleic acid encoding an E1A gene product, expressing the E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, where the nucleic acid is introduced by an adenovirus or a liposome, where the E1A gene product is an mini-E1A gene product, a method for suppressing growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product, where the nucleic acid is introduced in an adenovirus or a liposome, and a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug, where the chemotherapeutic drug is an alkylating agent, plant alkaloid, antibiotic or antineoplastic agent, where certain dependent claims state specific chemotherapeutic agents.

Claims 1-54 of '356 are drawn to a method for suppressing growth of a tumor comprising a neu overexpressing cell comprising introducing a nucleic acid encoding a mini-E1A gene product using a DNA/liposome complex and contacting the cell with a chemotherapeutic agent.

Therefore, at the time of the present invention, it would have been obvious to the ordinary artisan to arrive at the claimed methods for suppressing growth of a tumor as in claims 76, 106-121,150-172, 174-183 and 185-187 given claims 1-54 of '356.

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. 5,641,484 ('484) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J. Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior

inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'484 teaches methods of suppressing the growth of a neu oncogene mediated tumor comprising administering to said tumor a nucleic acid encoding an adenovirus E1A, and compositions of a DNA sequence encoding an adenoviral E1a protein complexed to a liposome (col. 24, lines 11-15 and col. 42, lines 50-59). The E1A gene product is taught to be an E1A 12S or 13S gene product (col. 2, lines 53-59).

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibits tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by contacting the cell in the tumor with a nucleic acid encoding an E1A gene

product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. U.S. Patent No. 5,643,567 ('567) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J. Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the

invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'567 teaches the suppression of the neu-oncogene mediated transformed or tumorigenic phenotype by the administration of a DNA sequence encoding an E1A gene product (col. 26, lines 15-22; col. 45, lines 4-14). The E1A gene product is described as either an 12S or 13S gene product (col. 2, lines 53-62).

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibit tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and

to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. 5,651,964 ('964) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al. (1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J. Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'964 teaches the suppression of neu-oncogene mediated transformation and tumor genesis by the administration of a DNA sequence encoding an E1A

gene product into tumor cells and an inhibition of the metastasis potential of the tumor cells (col. 18, lines 6-51).

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibit tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. 5,814,315 ('315) in view of Thatcher et al. (1989) Cancer 63, pp. 1296-1302, Powles et al. (1991) Brit. J. Cancer 64, pp. 406-410, Larsson et al. (1994) Cancer 74, pp. 2857-2862, Culine et al.

(1994) Eur. J. Cancer 30A, pp. 1239-1244, Norton (1994) Seminars in Oncol. 21, pp. 19-24, Valenti et al. (1993) Eur. J. Cancer 29A, pp. 1157-1161 and Chevalier et al. (1995) J. Clin. Oncol. 13, pp.314-322.

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'315 teaches the suppression of neu-oncogene mediated transformation and tumorigenesis by the administration of a DNA sequence encoding an E1A gene product into tumor cells and an inhibition of the metastasis potential of the tumor cells (col. 35, lines 6-51).

The art at the time of filing taught many anti-neoplastic, anti-cancer or anti-tumor agents. Thatcher teaches that melphalan is an anti-neoplastic agent, and therefor is a chemotherapeutic agent (page 1297, col. 1, lines 6-7). Powles teaches vincristine and mitomycin C inhibit tumor growth in patients with advanced breast cancer (page 407, col. 1, parag. 1). Larsson

teaches topisomerases II inhibitors such as daunorubicin and idarubicin are anticancer agents (page 2857, col. 2, parag. 1, lines 1-2). Culine teaches visblastine, dactinomycin, doxorubicin, bleomycin and cisplatin are chemotherapeutic drugs for the treatment of ovarian tumors (page 1240, col. 2, parag. 1). Norton teaches paclitaxel is an active anti-cancer agent in patients with stage IV breast cancer (page 21, col. 1, parag. 1, lines 1-5). Valenti teaches that VP16 and TNF are anti-tumor agents in mouse tumor models (page 1159, col. 2, parag. 2, lines 6-11). Chevalier offers motivation in stating that cytotoxic agents are often used in combination rather than alone (page 314, col. 2, lines 3-5).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. 6,197,754 ('754).

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the

reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'754 teaches methods to suppressing growth of a tumor comprising introducing an mini-E1A gene product to a tumor and where the method further comprises introducing a chemotherapeutic agent such as those specifically claimed in the present claims (col. 7, line 50 and col. 8, lines 25 and col. 28, line 52 to col. 29, line 2).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. 6,683,059 ('059).

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was

derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'059 teaches methods of suppressing tumor growth by administering a nucleic acid encoding a mini-E1A gene product, a vector comprising a nucleic acid segment encoding an mini-E1A gene products, a mammalian cell comprising the nucleic acid vector and a non-viral gene delivery complex comprising a mini-E1A gene product (col. 7, line 44 to col. 8, line 29). '059 further teaches the administration chemotherapeutic agents, the same as those presently claimed, in conjunction with the mini-E1A genes (col. 28, line 56 to col. 29, line 6).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Claims 76, 106-121 and 150-187 are rejected under 35 U.S.C. 103(a) as being obvious over U.S. Patent No. 6,326,356 ('356).

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art only under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 103(a) might be overcome by: (1) a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not an invention "by another"; (2) a showing of a date of invention for the claimed subject matter of the application which corresponds to subject matter disclosed but not claimed in the reference, prior to the effective U.S. filing date of the reference under 37 CFR 1.131; or (3) an oath or declaration under 37 CFR 1.130 stating that the application and reference are currently owned by the same party and that the inventor named in the application is the prior inventor under 35 U.S.C. 104, together with a terminal disclaimer in accordance with 37 CFR 1.321(c). For applications filed on or after November 29, 1999, this rejection might also be overcome by showing that the subject matter of the reference and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person. See MPEP § 706.02(l)(1) and § 706.02(l)(2).

'356 teaches methods of suppressing tumor growth by administering a nucleic acid encoding a mini-E1A gene product, a vector comprising a nucleic acid segment encoding an mini-E1A gene products, a mammalian cell comprising the nucleic acid vector and a non-viral gene delivery complex comprising a mini-E1A gene product (col. 7, line 44 to col. 8, line 29). '059 further teaches the administration chemotherapeutic agents, the same as those presently claimed, in conjunction with the mini-E1A genes (col. 28, line 56 to col. 29, line 6).

Thus it would have been obvious to the ordinary artisan at the time of filing to suppress the growth of a tumor comprising a neu oncogene cell by

contacting the cell in the tumor with a nucleic acid encoding an E1A gene product and a chemotherapeutic drug in amounts effective to suppress growth of the tumor, to suppress the growth of a neu-mediated cancer in an animal having or suspected of have the cancer comprising administering to the animal an effective combination of a nucleic acid encoding an E1A gene product and to make a pharmaceutical composition comprising a nucleic acid encoding an E1A gene product and a chemotherapeutic drug.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Deborah Crouch, Ph.D. whose telephone number is 571-272-0727. The examiner can normally be reached on M-Th, 8:30 AM to 7:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Amy Nelson can be reached on 571-272-0408. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Deborah Crouch, Ph.D.
Primary Examiner
Art Unit 1632

November 27, 2004